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HISTORICAL NOTE

Emil Heinrich Du Bois-Reymond (1818–96)

This distinguished physiologist is remembered for two dissimilar contributions: he was a founder of electrophysiology of nerve and muscle; and he described his own migraine.

Du Bois-Reymond first discovered that the peripheral passage of a nerve impulse was accompanied by an electrical discharge,[1] the action potential. After Matteucci, whose work he disparaged, he is often regarded as the founder of electrophysiology.

Du Bois-Reymond was born and studied medicine in Berlin. His father was a watchmaker in the Swiss canton Neuchatel but then moved to Berlin as a civil servant. Emil became a student of Johannes Müller, working with him from 1841 until Müller's death in 1858. After years of industrious study, he succeeded him to the Chair of Physiology. At that time he was in close contact with Helmholtz, Brücke, and Ludwig, liaisons that culminated in the foundation of a new institute of physiology in Berlin in 1877. Du Bois-Reymond was the chief for 20 years.

He was described in somewhat antiquated terms, a materialistic, and a mechanical physiologist. Du Bois-Reymond invented a refined sensitive nerve galvanometer and a stimulus producing induction coil. He showed an electric current in muscle (Müskelstrom). It was, he thought, owing to an attempt to preserve a resting (pre-existing) difference of potential between the negative inner and positive outer membranes of the fibre, which caused an electromotive force appearing with injury—an important and new concept:

> "The law of muscular current may be shortly expressed as follows: Any point of the natural or artificial longitudinal section of muscle is positive relation to any point of the natural or artificial transverse section . . . every particle of a muscle, however minute, ought to produce a current in the same manner as the whole muscle . . . As to the nerves . . . they are possessed of an electromotive power, which acts according to the same law as muscles."

He confirmed Matteucci's observation that during tetanus, the resting current flowing from an intact to an injured region is decreased and this negative variation was composed of a series of individual variations. This is called the negative variation (negative *Schwankung*) of Du Bois-Reymond. It now corresponds to variation in the action potential.

He investigated physiological tetanus[2] in 1850 and employed in his work the galvanometer. By this means he defined what he called electrotonus, the potential changes produced by an externally applied current; he also experimented with Faradic stimulation.

Du Bois-Reymond is also well known[3] for his views on the pathogenesis of migraine. Before his time, Robert Whytt had given an exposition of the spasm and relaxation of small blood vessels in migraine[4] that foreshadowed the vasospastic theories of Latham. Latham had initiated the vascular hypothesis and explained it as:

> "a contraction of the blood vessels of the brain, and so diminished supply of blood, produced by the excited action of the sympathetic; and that the exhaustion of the sympathetic following on this excitement causes the dilatation of the vessels and the headache."[5]

This topic seems remote from the electrophysiological work of Du Bois-Reymond, but his account in 1860 displays a personal stake:

> "a Tetanus takes place in the muscular coats of the vessels of the affected half of the head; in other words a Tetanus of the cervical portion of the sympathetic."[6] . . . every three or four weeks I am liable to an attack. I wake with a general feeling of disorder, and a slight pain in the region of the right temple, which, without overstepping the midline, reaches its greatest intensity at midday; towards evening it usually passes off. While at rest the pain is bearable, but it is increased by motion to a high degree of violence . . . It responds to each beat of the temporal artery. The latter feels on the affected side, like a hard cord, while the left is in normal condition. The countenance is pale and sunken, the right eye small and reddened . . . There may be left behind a slight gastric disorder; frequently, also, the scalp remains tender at one spot the following morning . . . For a certain period after the attack I can expose myself with impunity to influences which before would infallibly caused an attack."

This account is of interest showing the remission after an attack; and enophthalmos or miosis, and the red eye, which we would associate with cluster headache. He also deduced both the vascular component of the headache phase, and the role of the sympathetic nerves in inducing vascular constriction. Later, in 1873, Edward. Liveing in his classic text[7] accepted that dilatation of the arteries might explain the headache, but like Gowers, Liveing rejected the vascular theory as explanation of the varied content of the aura, its bilaterality in certain patients, the vegetative symptoms throughout the body, and the changes in patterns of attacks. Liveing regarded it as a "nerve storm."

> "a form of centrencephalic seizure, the activity of which is projected rostrally upon the cerebral hemispheres, and peripherally via the ramifications of the autonomic nervous system . . ."

In explaining his nerve storm theory[8] Liveing considered migraine along with other "neuroses" to be:

> "a primary and often hereditary disposition of the nervous system itself; this consists in a tendency to the irregular accumulation and discharge of the nerve force . . ."

Time has shown that Liveing's and Gowers' theories are closer to the truth than du Bois Reymond's, though we still do not understand the initial mechanism.

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